Targeting Topics: Recent Scientific References

Reviewed by Matthew Kohls

Nicotine-induced switch in the nicotinic cholinergic mechanisms of facilitation of long-term potentiation induction.

Yamazaki Y, Jia Y, Hamaue N, Sumikawa K
Eur J Neurosci 22(4):845-860, 2005

The authors investigated cellular mechanisms underlying improved cognitive function in Alzheimer's disease patients upon the administration of nicotine. To model Alzheimer's disease in rats, 2 μg of 192-IgG-SAP (Cat. #IT-01) was injected into the lateral cerebral ventricle. Examination of the lesioned animals suggests that nicotine promotes the induction of long-term potentiation by enhancing N-methyl-D-aspartate responses, and suppressing acetylcholine-mediated mechanisms in pyramidal cells.

Saporin and ricin A chain follow different intracellular routes to enter the cytosol of intoxicated cells.

Vago R, Marsden CJ, Lord JM, Ippoliti R, Flavell DJ, Flavell SU, Ceriotti A, Fabbrini MS
FEBS J 272(19):4983-4995, 2005

Some bacterial toxins such as Pseudomonas aeruginosa exotoxin A carry a KDEL-like C-terminal peptide sequence, which targets the protein to the endoplasmic reticulum. Saporin (Cat. #PR-01) is a plant ribosome-inactivating protein, which does not contain a KDEL-like sequence. Here the authors examined the intracellular pathways utilized by saporin. Although ricin, another plant ribosome-inactivating protein, could be visualized in the Golgi complex, saporin was not. The data suggest that saporin may utilize endosomes during its journey through the cell.

Selective acetylcholine and dopamine lesions in neonatal rats produce distinct patterns of cortical dendritic atrophy in adulthood.

Sherren N, Pappas BA
Neuroscience 136(2):445-456, 2005

In this work the authors examined lesions of acetylcholine afferents in 7-day-old rat pups, and the effect on dendritic development. 600 ng of 192-IgG-SAP (Cat. #IT-01) were administered to the ventricles of test animals. Various morphological changes in the retrosplenial cortex were observed, including smaller apical tufts and fewer basilar dendritic branches in layer V medial prefrontal cells. The data demonstrate that ascending acetylcholine afferents are very important in the development of cortical cytoarchitecture.

Estrogen contributes to structural recovery after a lesion.

Saenz C, Dominguez R, de Lacalle S
Neurosci Lett 392(3):198-201, 2006

The authors evaluated the trophic effects of 17ß-estradiol (E2) on cholinergic neurons of the basal forebrain after lesioning with 192-IgG-SAP (Cat. #IT-01). Ovariectomized female rats received 200 nl of 0.075 mg/ml 192-IgG-SAP followed by a subcutaneous pellet of E2, which was released over 60 days. Dendritic size in ovariectomized rats receiving the E2 was the same as in control animals, while ovariectomized rats receiving a placebo displayed a significant reduction in dendritic arborization.

Sleep-disordered breathing after targeted ablation of preBotzinger complex neurons.

McKay LC, Janczewski WA, Feldman JL
Nat Neurosci 8(9):1142-1144, 2005

Sleep-disordered breathing is common in elderly humans as well as patients with neurodegenerative disease. The authors investigated the role of preBötzinger complex neurons of rats in respiratory rhythm generation. Using the fact that preBötzinger complex neurons in the ventrolateral medulla express the neurokinin-1 receptor, animals were given bilateral injections of SP-SAP (Cat. #IT-07). Beginning 7 days post-injection, lesioned animals displayed marked respiratory disturbances during both sleep and wakeful periods.

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